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Time course of echocardiographic and electrocardiographic parameters in myocardial infarct in rats

AMARILDO MIRANDA[1**], RICARDO H. COSTA-E-SOUSA[1**], JOÃO P.S. WERNECK-DE-CASTRO[1], ELISABETE C. MATTOS[2], EMERSON L. OLIVARES[1], VANESSA P. RIBEIRO[1], MÁRCIA G. SILVA[1], REGINA C.S. GOLDENBERG[1] and ANTÔNIO C. CAMPOS-DE-CARVALHO[1]

[1]Laboratório de Eletrofisiologia Cardíaca, Bloco G2, Instituto de Biofísica Carlos Chagas Filho, CCS-UFRJ, Ilha do Fundão, 21949-900 Rio de Janeiro, RJ, Brasil
[2]Ecodata Exames Médicos Ltda., 22020-120 Rio de Janeiro, RJ, Brasil

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ABSTRACT

In animal models the evaluation of myocardial infarct size *in vivo* and its relation to the actual lesion found *post mortem* is still a challenge. The purpose of the current study was to address if the conventional electrocardiogram (ECG) and/or echocardiogram (ECHO) could be used to adequately predict the size of the infarct in rats. Wistar rats were infarcted by left coronary ligation and then ECG, ECHO and histopathology were performed at 1, 7 and 28 days after surgery. Correlation between infarct size by histology and Q wave amplitude in lead L1 was only found when ECGs were performed one day post-surgery. Left ventricular diastolic and systolic dimensions correlated with infarct size by ECHO on day 7 post-infarction. On days 7 and 28 post-infarction, ejection indexes estimated by M-mode also correlated with infarct size. In summary we show that conventional ECG and ECHO methods can be used to estimate infarct size in rats. Our data suggest that the 7-day interval is actually the most accurate for estimation of infarct size by ECHO.

Key words: myocardial infarction, rats, electrocardiogram, echocardiogram, infarct size.

INTRODUCTION

Cardiovascular diseases are the main cause of death and incapacity to work in the world (McFate 1985, Bristow 1994, Laurenti et al. 2000, Paul et al. 1994). Ischemic heart disease when not fatal leads to heart failure. With improvements in the treatment of myocardial infarction (MI), death from MI has declined but the incidence of heart failure has been growing steadily (Mann 1999, Mann and Taegtmeyer 2001). Left ventricular (LV) remodeling after MI plays a major role in the progression to heart failure (Mann 1999, Mann and Taegtmeyer 2001). Elucidating the mechanisms responsible for preventing and/or reversing the process of LV remodeling is one of the most important areas of investigation in cardiology. Interventions that have been shown to decrease mortality in patients with heart failure have had a favorable impact on the LV remodeling process (Greenberg et al. 1995, Doughty et al. 1997, Asahara et al. 1999, Khattar et al. 2001, Kocher et al. 2001, Orlic et al. 2001).

To better understand the pathophysiology and to develop new treatment regimens for heart diseases, investigators developed animal models that simulate human disease. The rat has been widely used as an experimental model for the understanding of the pathophysiology and management of myocardial infarction since 1954 (Johns and Olson 1954, Selye et al. 1960, Fishbein et al. 1978, Pfeffer et al. 1979, Fletcher et al. 1981, Hochman and

*Member Academia Brasileira de Ciências
**These authors contributed equally to this work.
Correspondence to: Antônio Carlos Campos de Carvalho
E-mail: acarlos@biof.ufrj.br

Bulkley 1982, Raya et al. 1988, Coudray et al. 1993, Nguyen et al. 2001). However, after 50 years and even with the more recent use of imaging techniques such as echocardiography (De Simone et al. 1990, Baily et al. 1993, Litwin et al. 1994, 1995), at present the evaluation of myocardial infarct size in vivo and its relation to the actual lesion found in *post-mortem* histopathology is still a challenge.

The specific purpose of the current study was to address the following questions: Can the routine electrocardiogram (ECG) and/or the echocardiogram (ECHO) predict adequately the extension of the infarct in the rat? How do these exams change with time after infarction? Is there a correlation between infarct size measured by histopathology and alterations in these exams?

MATERIALS AND METHODS

This investigation follows the guidelines proposed in the *Guide for the Care and Use of Laboratory Animals* published by the US National Institutes of Health (NIH Publication No. 85-23, revised 1996) as attested by the competent institutional board.

Animals

Female inbred Wistar rats (173 to 227g) were obtained from the Centro de Pesquisa Gonçalo Muniz (Fiocruz-Bahia/Brazil). Animals were housed at controlled temperature (23°C) with daily exposure to a 12h light-dark cycle (lights on 08:00-20:00 h) and free access to water and standard rat chow.

Myocardial Infarction

Rats were anesthetized by intra peritoneal injection of ketamine hydrochloride (50 mg/kg) and xylazine (2 mg/kg). The same anesthetic protocol was used in every exam when animal manipulation was necessary. The technique for production of myocardial infarction has been described (Johns and Olson 1954, Selye et al. 1960) and was used here with minor modification. After adequate anesthesia, animals were placed in supine position and ventilated manually. Left anterior thoracotomy was performed at the fourth intercostal space and the heart was rapidly exteriorized, and a 6-0 silk suture was tightened around the proximal left anterior descending coronary artery. Positive end-expiratory pressure was applied to fully inflate the lungs, and the muscle layer and skin were closed together, closing the thorax. Sham-operated rats (sham) underwent identical surgery but the suture was not tightened around the coronary artery.

Electrocardiographic Study

Rats anesthetized with ketamine and xylazine, as described before, were placed in the supine position. The ECG was recorded with a conventional electrocardiograph (Cardimax FX-2111 – Fukuda Denshi, Japan) to record the classic limbs leads (L_1, L_2, L_3, aVR, aVL, aVF) at 50 mm/sec and 20mV/mm. All animals were rigorously maintained in the same position to obtain consistent direction and magnitude of cardiac vectors from limb-lead scalar electrocardiograms. The ECG parameters studied were: heart rate, P wave amplitude (mV), PR interval (ms), QRS duration (ms), QT interval (ms), frontal QRS axis (ÂQRS), QRS amplitude index (I-QRS, the sum of absolute QRS complex voltage in L_1, L_2 and L_3 in mV) and presence of Q wave in L_1, L_2, L_3 and aVF. All amplitude and interval measurements were performed in lead L_2. ÂQRS was measured using L_1 and AVF leads.

Echocardiographic Study

In this study we used an echocardiographic color-system (Megas/Esaote, Italy) equipped with a 10MHz electronic-phased-array transducer. Under intraperitoneal ketamine and xylazine anesthesia, as described before, the chests of the animals were shaved and they were maintained either in left lateral decubitus or supine position. Images were obtained from the left paraesternal and apical window. Short-axis 2-dimensional views of the left ventricle (LV) were taken at the level of the papillary muscles to obtain the M-mode recordings (De Simone et al. 1990, Baily et al. 1993, Litwin et al. 1994, 1995). Anterior and posterior end-diastolic and end-systolic wall thicknesses and LV, left atrium and aorta internal dimensions were measured following the American Society of Echocardiography (ASE) leading-edge method. The systolic function was expressed by the fractional shortening (SF%) or ejection fraction (EF%) obtained from the M-mode tracings or by ejection fraction estimated by Simpson's method. The pulsed-wave Doppler spectra of mitral flow was recorded from the apical four-chamber view with the guidance of the

color Doppler. All Doppler spectra (mitral flow velocity pattern: peak early diastolic filling velocity, E velocity; peak filling velocity during atrial contraction, A velocity; and their E/A ratio) were recorded and morphologic parameter values were measured during the echocardiographic exam.

Post-mortem Histopathological Analysis

The rats were sacrificed on days 1, 7 and 28 post-surgery and a complete necropsy was performed. The hearts were removed, weighted and their weight was corrected by the body weight of the animal. The ventricles were then cut in four slices from apex to base. The slices had approximately the same thickness (1-2 mm) and were named slices A, B, C and D from the cardiac apex to base, respectively (Spadaro et al. 1980). Histological analysis with hematoxilin-eosin and picrosirius staining was performed in representative sections obtained from each slice using an Axiovert 100 microscope (Zeiss Inc – Germany). Sections stained with picrosyrius were recorded with a digital camera and stored for posterior analysis. The digital files were analyzed with the ImageJ software (version1.27z, National Institute of Health, U.S.A.) which allowed us to measure the infarct size of the left ventricle (LV). The method used to measure the infarct size has been described in the literature (Spadaro et al. 1980). It can be summarized as follows: sections from each slice (A, B, C and D) were evaluated individually. The length of the infarcted endocardium is measured, as well as, the total perimeter of the endocardial surface. From the ratio of these values, the percentage value of infarcted endocardium is calculated. The same procedure is done for the epicardial surface, obtaining the value of the percentage of infarcted epicardium (Spadaro et al. 1980). From these two values the average percent infarct size was estimated. This process is carried out in slides from each one of the four slices and, in the end, the percentage of infarcted LV of the heart analyzed is calculated from the mean values obtained from the four slices (Spadaro et al. 1980).

Statistical Analysis

All statistical analysis was performed with Statistica™ program version 6.0 (Stat Soft, Inc; EUA). Values are expressed as mean ± standard deviation (SD), except in graphs, where the values are usually expressed as mean ± standard error (SE). To compare two experimental groups, Student's *t* test was used. More than two groups were compared with ANOVA test and group differences determined with the Tukey procedure. Spearman rank test was used for correlations. Differences were considered significant if $p < 0.05$.

RESULTS

We compared three experimental groups: normal, infarcted and sham operated rats, following the time course of ECGs, ECHOs and histopathology for up to 28 days.

Electrocardiography

No significance difference was found in ECG parameters between sham (N = 39) and normal (N = 50) rats. In contrast, important alterations were found in the ECG of the infarcted group (N = 84). When compared to sham and normal rats, all infarcted animals showed the presence of Q wave in lead 1, with variable amplitude. This finding was present from the first day after infarction and remained until the end of the observation period (28 days post-surgery), independent of infarct size. Q wave amplitude in L1 was larger on day 1 post-infarct (0.33 mV ± 0.11; $p < 0.001$), decreased on day 7 (0.18 mV ± 0.07) and remained constant until the end of experiment (28 day) (0.17 mV ± 0.07). Most Q waves were present as QS waves and in the few ECGs where R waves could be found in L1 lead their amplitudes were extremely low.

Infarcted animals showed a marked rightward deviation of the QRS axis when compared to sham-operated rats (+133° ± 26 *vs.* +66° ± 11; $p < 0.001$; infarcted *vs.* sham operated, respectively; at day 1). Again, this alteration was present in all infarcted animals, independent of infarct size, but not in sham or normal rats. The axis deviation was greater on day 1 post-infarct and then decreased (ÂQRS = 133° ± 26 at day 1; 118° ± 6 at day 7; 117° ± 7 at day 28; $p < 0.05$; day 7, 28 *vs.* day 1), but at the end of the experiment the axis was still deviated. Moreover, reduction in QRS amplitude index was observed in the infarcted group (0.878 ± 0.304 mV *vs.* 1.466 ± 0.199 mV; $p < 0.001$; infarcted *vs.* sham operated, respectively) with its value progressively decreasing with time. There was no statistical difference in PR interval (44 ms ± 10 *vs.* 48 ms ± 9; infarcted *vs.* sham operated, respectively), duration of QRS (20 ms ±

5 *vs.* 21 ms ± 4; infarcted *vs.* sham operated), P wave duration in L2 (25 ms ± 8 *vs.* 26 ms ± 8; infarcted *vs.* sham operated, respectively), QT interval (87 ms ± 8 *vs.* 85 ms ± 10; infarcted *vs.* sham operated) and heart rate (HR = 294 bpm/min ± 42 *vs.* 277 ± 35; infarcted *vs.* sham operated) between groups, at all time intervals studied. The P wave amplitude was greater in the infarcted group (0.082 mV ± 0.029 vs 0.075 mV ± 0.024; $p < 0.01$; infarcted *vs.* sham operated, respectively) at all time intervals.

Echocardiography

The main echocardiographic parameters measured are shown in Table I. Although heart rate can influence significantly echocardiographic measurements, in our study BR was not different between groups. Due to the use of different echocardiographic techniques to evaluate systolic function in the literature, we decided to measure left ventricular systolic function variations between groups using ejection fraction determined by Simpson's method, and shortening and ejection fraction determined by M-mode recording. All three indexes of systolic function were significantly decreased in infarcted rats when compared to normal or sham-operated animals. Time course of the impairment in systolic function after infarction can be seen in Figure 1 as determined by the three methods. In contrast to the continuously decreasing values observed in the infarcted animals, sham operated rats maintained constant values for systolic function during the whole observation period.

As expected, aorta diameter (Ao) was not altered between groups. However, left atrium diameter increased in the infarcted group ($p < 0.001$, Table I), leading to the increased La/Ao relation ($p < 0.001$) shown in Table I. There was also a significant increase in LV diastolic (LVDd) and systolic (LVDs) dimensions in infarcted as compared to sham hearts already at 1 day after coronary ligation ($p < 0.001$). Thereafter, a further increase in these dimensions was seen at 7 and 28 days (Table I).

Anterior left ventricular wall thickness was significantly reduced in MI hearts during systole and dyastole at 1 day post-surgery ($p < 0.001$) and remained unchanged after 28 days, while the posterior wall thickening during systole decreased significantly only at 28 days in post-MI hearts ($p < 0.01$, Table I).

E/A ratio increased in the infarcted group at 7 and 28 days (Table I) revealing the restrictive pattern during diastolic filling of the infarcted heart.

Necropsy, Histological Examination and Infarct Size

The necropsy showed an increase in cardiac mass (at 28 days post-surgery) when corrected to body weight in the MI group (N = 19) as compared with sham-operated rats (N = 9) ($5.07 \times 10^{-3} \pm 1.16 \times 10^{-3}$ *vs.* $4.05 \times 10^{-3} \pm 3.53 \times 10^{-3}$; $p < 0.05$). Infarct size determined by planimetry in 27 hearts was 38% ± 11. Slice A (apex) presented the largest infarction area (51% ± 15), followed by slice B (43% ± 14), slices C and D (41% ± 10; 19% ± 22). There was no statistical difference in the infarct size when comparing infarcted animals sacrificed on day 1 (n = 32), 7 (n = 27) or 28 (n = 19) post-surgery.

The histological examination of the infarcted myocardium, performed 1 day after the injury, showed an extensive area of coagulation necrosis with hemorrhage in the subepicardium. Careful examination, at high magnification, revealed marked capillary stasis, several vascular lacunae with endothelial discontinuities, expanded extracellular spaces, optically empty or filled by erythrocytes and infiltration of the vascular walls by many polymorphonuclear leukocytes. Since picrosyrius does not stain the heart 1 day post-infarction, we used the presence of histological alterations described above to estimate infarct size at this time point. Seven days after the injury, a dense acute inflammatory infiltrate was evident, starting at the periphery of the damaged area and composed mainly by polymorphonuclear leukocytes and macrophages. Borders of the injured area were always clearly identifiable. The repairing process was completed within 28 days, with formation of a dense paucicellular fibrous scar.

Correlation Between Measured Variables in Infarcted Animals

There was a weak correlation (R = 0.47; p = 0.01; N = 27) between infarct size measured by planimetry and Q wave amplitude in lead L1 of ECGs performed on day 1 post-surgery but no correlation was found between these parameters at later time points. Only in the echocardiographic exams performed on day 7 post-infarction, left ventricular diastolic (LVDd) and systolic (LVDs) dimen-

TABLE I

Echocardiographic parameters in experimental groups. HR = Heart rate; Ao = Aorta diameter; La = Left atrium diameter; LVDs = Left ventricular diameters at systole; LVDd = Left ventricular diameters at diastole; AWTd = Anterior wall thickness in diastole; AWTs = Anterior wall thickness in systole; PWTd = Posterior wall thickness in diastole; PWTs = Posterior wall thickness in systole. Values expressed in mean ± SD (standard deviation); Infarcted *vs.* Sham-operated: *p < 0.05, **p < 0.01, ***p < 0.001.

	Experimental Groups											
	Sham-operated						Infarcted					
	1 day		7 days		28 days		1 day		7 days		28 days	
	N	Mean±SD	N	Mean ±SD	N	Mean±SD	N	Mean ±SD	N	Mean±SD	N	Mean ±SD
HR (bat/min)	12	286±34	11	259±51	9	236±50	26	283±32	26	253±42	18	249±37
Ao (mm)	12	3.4±0.1	11	3.4±0.2	9	3.5±0.2	26	3.2±0.3	26	3.2±0.3	18	3.3±0.3
La (mm)	12	3.1±0.3	11	3.5±0.2	9	3.6±0.3	26	3.7±1.1	26	4.1±0.8**	18	4.5±1.1*
La/Ao	12	0.92±0.09	11	1.00±0.05	9	1.03±0.08	26	1.14±0.28*	26	1.28±0.27**	18	1.35±0.46**
LVDd (mm)	12	5.6±0.6	11	6.3±0.6	9	6.2±0.5	26	6.8±0.7***	26	8.0±0.5***	18	8.5±0.9***
LVDs (mm)	12	2.6±0.9	11	2.5±0.6	9	2.4±1.0	26	5.0±0.9***	26	6.0±0.8***	18	6.7±1.0***
AWTd (mm)	12	0.83±0.08	11	0.81±0.03	9	0.91±0.08	26	0.73±0.07***	26	0.79±0.06*	18	0.82±0.07**
AWTs (mm)	12	1.52±0.45	11	2.12±0.16	9	1.98±0.62	26	0.74±0.07***	26	0.89±0.35***	18	0.83±0.08***
PWTd (mm)	12	0.84±0.08	11	0.84±0.05	9	1.20±0.83	26	0.82±0.06	26	0.86±0.05	18	0.90±0.06**
PWTs (mm)	12	1.98±0.19	11	2.31±0.19	9	2.35±0.22	26	1.97±0.29	26	2.05±0.23	18	2.02±0.30**
E velocity (cm/s)	12	59.1±8.6	11	58.5±4.4	9	56.2±5.5	26	55.4±9.8	26	65.4±11.0*	18	66.4±17.1*
A velocity (cm/s)	12	34.2±7.2	11	36.5±5.9	9	36.2±4.2	22	26.2±5.2***	26	26.9±10.5**	18	34.6±14.2
E/A ratio	12	1.76±0.28	11	1.63±0.23	9	1.56±0.18	22	2.24±1.02	26	3.00±1.71**	18	2.70±2.69*

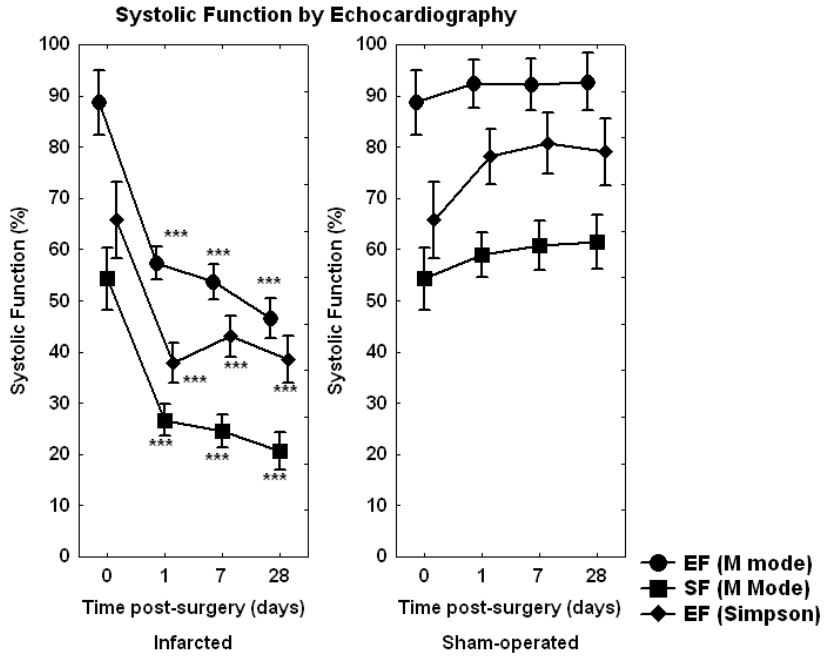

Fig. 1 – Time course of systolic function in infarcted and sham-operated rats. All infarcted rats have systolic function decreased independently from the measurement method used. EF = ejection fraction; SF = shortening fraction. Day 0 is before surgery (normal). Values expressed as mean ± SE (standard error). Infarcted *vs.* Sham-operated: ***p< 0.001.

sions correlated with infarct size (R = 0.59, $p < 0.05$, N = 22; R = 0.71, $p < 0.001$, N = 22, respectively) as shown in Figures 2-A and 2-B. Importantly, on day 7 post-infarction, there was a negative correlation between infarct size, ejection and shortening fraction determined by M-mode as shown in Figures 2-C, 2-D. Negative correlation was also found between infarct size, ejection and shortening fraction on day 28 post-surgery (R = –0.63, $p < 0.05$, N = 11; R = –0.65, $p < 0.05$, N = 11; respectively) and There is correlation between E/A ratio and infarct size at 28 days post infarction (R = 0.71, N = 10, p = 0.02). This implies that M-mode echocardiography can be used as an estimate of infarct size in this time interval.

DISCUSSION

In the present work, we studied the time course of electrocardiographic and echocardiographic alterations in rats submitted to MI surgery. Our observations spanned the acute (1 day) and chronic phases of the infarct (28 days) allowing a critical evaluation of the advantages and limitations of the different exam modalities, always comparing the results with histopathology, which is considered the "golden standard" in a study like this.

ECG alterations in the infarcted group were evident; most of them being present in the first day after surgery and remaining unaltered thereafter. QRS amplitude index decreased in the infarct group. The decrease of this index with time most probably results from progressive loss of myocytes and consequently their substitution by fibrous tissue (electrically inactive) that occurs after the infarction, as found in other studies (Santos and Masuda 1991). In humans the decrease in QRS index is also attributed to expansion of the fibrous scar (De Serro-Azul et al. 1974). In our study no correlation was found between QRS index and infarct size.

ÂQRS was another ECG parameter altered in the infarcted group. All infarcted rats had angles greater than 92 degrees and this alteration was present from the first day post-infarction until the end of the experiment. The marked rightward deviation of the QRS axis is due to the loss of LV mass with or without RV hypertrophy

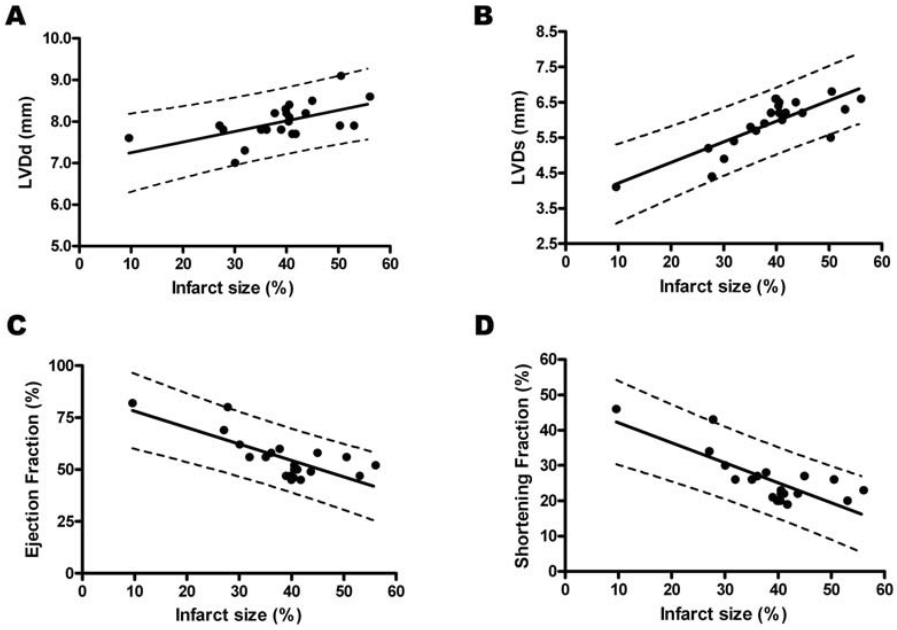

Fig. 2 – **A.** Correlation between infarct size and left ventricular diastolic diameter (LVDd) measured at 7 days post-surgery. $R = 0.59$, $p < 0.05$, $N = 22$. **B.** Correlation between infarct size and left ventricular systolic diameter (LVDs) measured at 7 days post-surgery. $R = 0.71$, $p < 0.001$, $N = 22$. **C.** Correlation between infarct size and ejection fraction (%) measured by M-Mode at 7 days post-surgery. $R = –0.57$, $p = 0.003$, $N = 21$. **D.** Correlation between infarct size and shortening fraction (%) measured by M-Mode at 7 days post-surgery. $R = –0.59$, $p = 0.004$, $N = 21$. Doted lines delimit 95 % confidence interval.

(our data show rightward deviation of QRS as soon as 1 day post-surgery, at a time when RV hypertrophy could not have happened). The presence of pathological Q wave in L1 results from myocardial necrosis, localizing the infarct lesion to the left lateral wall of the heart (De Serro-Azul et al. 1974, Norman and Coers 1960). Q wave amplitude measured on day 1 post-surgery and infarct size correlated, suggesting that ECGs recorded at this time could give a rough estimate of infarct size. At later time points, 7 and 28 days post MI, we found no correlation between Q wave amplitude and infarct size as reported in the literature (Bonilha et al. 2005). In contrast to reports by other authors (Santos and Masuda 1991, Tseng et al. 1995), in our study the infarcted rats did not have alterations in PR interval or QRS duration in L2. Overall, the conventional ECG is an excellent marker for MI but a poor predictor of infarct size.

The echocardiogram is a useful tool in the evaluation of cardiac function and alterations in the morphology of the heart in a noninvasive form. ECHO evaluation showed that infarcted animals had ventricular dysfunction. An increase in the left atrium dimensions and of La/Ao relation was clearly observed at 7 days post-infarction (Table I) progressing with time. These findings are similar to those found in the literature (Raya et al. 1988, Litwin et al. 1994, Palojoki et al. 2001, Gaballa and Goldman 2002) and reflect the hemodynamic alterations occurring after the infarction as a consequence of the failure of the LV and its consequent dilatation leading to the overload of the left atrium. It is interesting to observe that these values were already modified within 24 hours of the infarction and the ECHO was sensitive enough to detect these alterations precociously.

It is noteworthy that systolic function of the infarcted rats showed no improvement during the time course of the experiments. The evolution of the infarction process led to a continuous cardiac remodeling. Cardiac remodeling was present in all the animals of the infarcted group, being characterized mainly by progressive dilatation of the LV, reduction of the thickness of the in-

farcted LV wall, increase of the total cardiac mass and increase of the left atrium chamber. By ECHO we found a correlation between infarct size and LV dilatation only at 7 days post MI. A negative correlation between infarct size and ejection fraction and shortening fraction by M-mode was found at 7 and 28 days post MI (Figs. 2-C and 2-D).These data suggest that the best time point to estimate infarct size by M-mode ECHO is at 7 days post-surgery.

Three patterns of ventricular filling, employing Doppler, have been used to describe diastolic alterations (Prunier et al. 2002). These three patterns can be found in MI in rats, being related to the severity of the cases (Litwin et al. 1995, Prunier et al. 2002). The exact value of the E/A ratio to classify the filling pattern as restrictive is controversial. Each laboratory should establish its proper standard value for this parameter, considering the equipment and anesthetic protocol used in the experiment. Prunier et al. (2002) considers values of E/A ratio above 2.0 as restrictive, while Olivares et al. (2004) consider restrictive values, those above 3.0. In this work only one animal in the sham-operated group presented values of E/A $>$ 2.0, but this value did not reach 2.5. Our normal group was not different: only one animal presented E/A $>$ 2.0, however not exceeding 2.4. In our infarcted group values of E/A between 1.0 and 2.0 (included) were found in 52% of the examinations. The other 48% had values above 2.0. With time the E/A ratio in the infarcted group increases, being maximal at 28 days post-infarct, when animals with E/A ratios of up to 8.0 were observed, showing a strong trend to the restrictive pattern. Thus, in the examinations at 28 days post-infarct, a positive correlation between E/A ratio and infarct size was found, suggesting that animals with a larger infarct have greater diastolic dysfunction. These findings imply that besides systolic dysfunction, the infarcted group also has important diastolic dysfunction.

In summary our data show that ECG and ECHO detect non-invasively MI in rats and that these methods can be used to estimate infarct size. The most significant correlations were found between echocardiographic parameters and infarct size as measured by histopathology; among these, LV diastolic and systolic volumes 7 days after MI, and M-mode SF% and EF% at 7 and 28 days post-MI. Our data further suggest that the 7 day interval is actually the most accurate for estimation of infarct size by echocardiography.

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RESUMO

Nos modelos animais a medida do tamanho do infarto do miocárdio "in vivo" e sua relação com o tamanho da lesão encontrada no exame "pos-mortem" ainda é um desafio. A finalidade do presente estudo é verificar se um eletro (ECG) e ecocardiograma (ECO) rotineiros poderiam ser utilizados para predizer a extensão do infarto em ratos. Ratos Wistar foram infartados pela ligadura cirúrgica da artéria coronária descendente anterior e exames eletro, ecocardiográficos e histopatológicos foram realizados 1, 7 e 28 dias pós-infarto. Foi encontrada correlação entre o tamanho do infarto medido pela histopatologia e a amplitude da onda Q em D1 apenas nos ECGs realizados no primeiro dia após a cirurgia. Os diâmetros da cavidade ventricular esquerda medidos em sístole e em diástole pelo ECO correlacionaram-se com o tamanho do infarto no sétimo dia pós-infarto. Ainda mais, no sétimo e vigésimo oitavo dias pós-cirurgia, os índices sistólicos estimados pelo Modo M também se correlacionaram com o tamanho do infarto. Em resumo, nós mostramos que um ECG e ECO convencionais são capazes de estimar a extensão do infarto do miocárdio em ratos. Nossos dados sugerem que o tempo mais adequado para estimar o tamanho do infarto pelo ECO é 7 dias pós-cirurgia.

Palavras-chave: infarto do miocárdio, ratos, eletrocardiograma, ecocardiograma, tamanho do infarto.

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